THE SURGICAL TREATMENT OF HEPATIC ASCITES.

AUSTIN O'MALLEY, M.D., PH.D., LL.D.,
PATHOLOGIST TO ST. AGNES' HOSPITAL, PHILADELPHIA.

To have a knowledge of the surgical treatment of hepatic ascites we should examine the facts and theories—and the latter predominate—which are connected with the etiology of hepatic portal cirrhosis; and at the outset we find that each writer upon the hepatic cirrhoses is inclined to offer a new classification of these conditions. Lancereaux[1] and Hawkins[2] arrange the cirrhoses as alcoholic, malarial and syphilitic; Chauffard[3] has a very complicated grouping[4] which is founded on the supposed causes of the cirrhoses; Adami,[5] Schiassi,[6] and Rolleston[7] group the cirrhoses for anatomical reasons; Senator offers another category; we have the French minute divisions, and so on.

There are two groups which comprise all: the portal cirrhoses and the biliary cirrhoses. In portal cirrhosis we find the hobnailed liver; hæmatemesis is common and it is observed early among the symptoms; ascites usually appears at the end of the disease, although death from toxæmia may occur without ascites; jaundice is not a marked characteristic, and whether the liver is large or small there is marked fibrosis; there is hypertrophy of the spleen, but this symptom is more prominent in biliary cirrhosis; the multilobular fibrosis consists in groups of lobules within a fibrous envelope, and the cirrhosis in the majority of cases is caused by toxines that are carried in by the portal vein. In biliary cirrhosis jaundice is constant and evident; hæmatemesis and ascites are rare; the liver is big and smooth and the fibrosis is monolobular or diffuse, or mixed; the spleen is large, often very large.

Portal hepatic cirrhosis is, in the general opinion, caused by toxines or toxine-producing agents, like micro-organisms. If the toxines come through the veins, by far the most frequented way, we find the typical portal form of cirrhosis; if it is brought in by the arteries the liver disease may approach the biliary kind.

The portal system may carry into the liver the results of alcoholic poisoning. Whether alcohol causes hepatic cirrhosis by direct action or indirectly is an unsettled question. Alcoholism frequently precedes hepatic cirrhosis, and it appears to lesson the resistance of the liver to sclerogenic poisons, but it is very doubtful that it directly causes cirrhosis. Formad[1] in 250 autopsies on drunkards that had died suddenly from alcoholic excess found hepatic cirrhosis in only six cases. Alcohol directly brings on only fatty degeneration of the liver; and although of great importance in the indirect etiology of some cases of hepatic cirrhosis, that importance is commonly exaggerated.

---

[1] Traité des maladies du foie et du pancreas, 1899.

[2] Allbutt's System of Medicine, vol. iv.

[3] Traité de médecine, 1892, tome iii. p. 827.

[4] Rolleston's Diseases of the Liver, 1905, p. 174.

[5] Sajous' Annual, 1899, vol. ii. p. 284.

[6] Di talune cirrosi epatiche e del loro trattamento, 1905.

[7] Op. cit., p. 176.

Arsenic probably has set up hepatic cirrhosis directly in certain cases; lead more probably does not; and experimentally in animals bacterial toxines have also induced cirrhosis, especially when these toxines had been introduced together with alcohol, but this question also is unsettled as regards man. We find tuberculosis associated with cirrhosis oftener than with any other disease, but this is due to impaired resistance which permits cirrhosis and also tuberculosis. As a rule the cirrhosis precedes the tuberculous infection. Tuberculosis associated with cirrhosis attacks the lungs oftenest, and next in frequency the peritoneum.

Kelsch and Keiner[2] describe a malarial hepatic cirrhosis. Schiassi[3] gives two cases of atrophic hepatic cirrhosis in patients suffering from chronic malaria with splenomegaly and ascites. Cardelli, Tommasi, and De Renzi have reported examples of atrophic malarial cirrhosis; and Galvagni, Ughetti, Mazzotti, and Finzi have found in malaria the liver reduced to a small soft mass, the" cirrosi flaccida" of Galvagni. On the other hand, American physicians do not find malarial hepatic cirrhosis. Osler[4] in nine years at Johns Hopkins Hospital did not see one case. Backer[5] thinks malarial affections of the liver are secondary to gastrointestinal and splenic disturbance; so does Chauffard[6]. The Italian pathologists incline to the opinion that toxines arising in the spleen induce hepatic cirrhosis, but the question is by no means settled.

The East Indian and Egyptian Mohammedans and the East Indian Brahmins, none of whom uses alcohol, frequently show cirrhosis of the liver. There is a biliary form very marked in Brahmin children which is prone to recurrence in the same family. Rolleston[7] says that no less than fourteen children of the same parents died of this disease. It is ascribed in these cases to intestinal intoxication after irritation by acetic, butyric, and lactic acids; and in Oriental adults the gastrointestinal irritation of spices and other stimulating articles of diet is thought to be the cause. These cases add no little weight to the supposition that hepatic cirrhosis can have a gastrointestinal source.

[1] Transactions of the Association of American Physicians, vol. i. p. 225.

[2] Archiv. de physiol. norm. et path., 1878, p. 571; 1879, p. 354.

[3] Di talune cirrosi epatiche e del loro trattamento, 1905.

[4] Practice of Medicine, 1901.

[5] Johns Hopkins Hospital Reports, vol. v. p. 241.

[6] La sem méd., 1899, p. 177.

[7] Diseases of the Liver, p. 187.

In the various forms of portal cirrhosis ascites becomes a grave complication. The majority of patients that die directly of cirrhosis have ascites. Ordinarily 50 per cent. of cirrhotic patients develop ascites, and when they die of cirrhosis directly the percentage is about 80.

The typical ascitic fluid in hepatic cirrhosis may be clear, yellowish, or somewhat green, or it may be slightly stained with bile. It has a sp. gr. of 1.008 to 1.015, and it is more albuminous than the fluid in renal ascites, and less albuminous than that of cardiac ascites. If the sp. gr. is above 1.015, the albumin above 0.4 per cent., and leukocytes are present, these qualities are indicative of peritonitis. It is difficult at times to differentiate the ascites arising from chronic peritonitis and that from cirrhosis. A constant rapid recurrence of the ascites after paracentesis is found in chronic peritonitis and not in cirrhosis. Ascites from tuberculous peritonitis is not seldom mistaken for cirrhotic ascites and *vice versa*. In tuberculous peritonitis the sp. gr. of the ascitic fluid commonly is about 1.020, it has more albumin than has the fluid from alcoholic cirrhosis, and it is turbid.

The ordinary medical treatment of cirrhosis is given in the standard text-books, but what may be done surgically to relieve ascites has not been set forth so clearly. The underlying fact in the surgical treatment of hepatic ascites is that the extravasation of serum into the abdominal cavity may commonly be averted by sending the venous blood through the veins of the belly wall instead of through the portal system. The portal system is made up of the portal, gastric, splenic, and the superior and inferior mesenteric veins, and these drain the entire alimentary canal below the gastric end of the œsophagus. Some think that the cause of ascites is obstruction of this drainage system in the cirrhosis of the liver. That such a condition does check the flow of blood through the liver is a fact, but it is not enough in itself necessarily to cause ascites. Ligation of the portal vein in animals does not bring on ascites; moreover, the onset of dropsy in man frequently is sudden, not gradual in proportion to the increase in the hepatic fibrosis. Probably ascites in the great majority of cases is a result of toxæmia, of peritonitis, of perihepatitis, or of cardiac failure. It is rarely caused by portal thrombosis or by the cirrhosis itself. The symptoms of drowsiness and multiple hemorrhages in renal toxæmia and in hepatic cirrhosis suggest toxæmia as a factor in the liver condition which induces a lymphagogic ascites. Chronic peritonitis in cirrhosis is often accompanied by ascites, but marked ascites may occur without peritonitis.

The theory that perihepatitis also causes ascites has been offered, but it is not established. Cardiac weakness may in cirrhosis give rise to ascites as a concomitant phenomenon rather than a direct effect of the liver condition.

Whatever are the facts involved in the etiology of hepatic ascites, and they are not at all clearly known, fixation of the omentum or the spleen, or both, to the belly wall has repeatedly removed the ascites by establishing a collateral circulation. In cases where the spleen is thought to be especially the source of the agents that bring on ascites, splenectomy has been tried. In the splenectomies for Banti's disease and somewhat similar conditions the mortality is about 25 per cent. under favorable circumstances, and if this operation is done in the ascitic stage the mortality is very high. The statistics as to improvement in health are not formulated, because, in most of the reported instances, if the patient is living a few weeks or months after the splenectomy, and the ascites has subsided, the operation is classed as successful. As early as 1889 Van der Meulen did epiplopexy to establish a collateral circulation through the belly wall and thus remove ascites. This was an outcome of a suggestion by Talma. Talma in 1898 also fixed the spleen to the belly wall after epiplopexy alone had not removed the ascitic condition.

Greenough[1] collected the statistics of 104 cases of cirrhosis in which an attempt to establish a collateral circulation had been made by epiplopexy or splenopexy or both, and in 57 per cent. of these cases there was no real improvement; in 42 per cent. there was improvement; in 9 per cent. the patients were living and in improved health two years after operation.

Lejars[2] also brought together statistics for 78 such cases. Thirty-six died soon after operation (46.15 per cent.); in 28 (35.89 per cent.) the ascites disappeared; and in 14 (17.94 per cent.) there was notable improvement.

Judging from final results alone there is slight difference between splenectomy, where it seems to be indicated, and epiplopexy with splenopexy, but the latter operation is more conservative and easier of performance; and since its results are at the least as good as splenectomy, probably somewhat better, it should be preferred. Despite the array of evidence and the ingenious theorizing, when all is sifted, either operation is very largely empirical; but in about one-third of the cases where fixation to the belly wall has been done the ascites has been relieved, and that is certainly very ample justification for the operation. Moreover, it may be done when splenectomy is contraindicated.

Schiassi,[3] of Bologna, reports the following cases of his own:

CASE I.—Hepatic cirrhosis with ascites. Schiassi holds that the cirrhosis was pericardiac in origin. He did epiplopexy in December, 1898. At present, more than six years later, the liver is big, hard, and painless; the spleen is small; there has been no trace of ascites.

---

[1] THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, vol. cxxiv. p. 979, December, 1902.

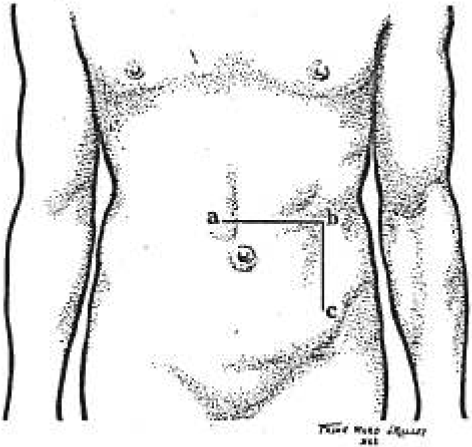
[2] La semaine médicale, March 25, 1903, p. 93.

[3] Di talune cirrosi epatiche e del loro trattamento, 1905.

CASE II.—Alcoholic atrophic cirrhosis with ascites. Epiplopexy in August, 1903. After eighteen months the liver functions were partly in action, the nutrition was good, and there was no trace of ascites.

CASES III. and IV.—Malarial metasplenic hepatic cirrhosis. He fixed both omentum and spleen, one in August, the other in November, 1902. The first case, a woman, had a decidedly atrophic liver, but she was in good condition otherwise. Before operation 100 litres of ascitic fluid had been withdrawn in various tappings. After operation a small quantity of fluid remained in the belly, but this has not increased in two years. The spleen is reduced very much. The second case, a man, has had no ascites for three years and his general condition is improved.

FIG. 1

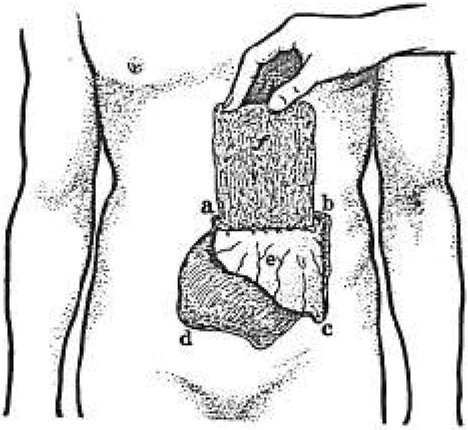


a, *b*, *c*, skin and muscle incision.

The fifth patient was a case of Banti's disease. The omentum and spleen were fixed in April, 1901. At present the spleen is reduced to two-thirds of the size it had at the time of the operation; there is no ascites; the hæmoglobin is 90 per cent.; the patient is well nourished.

There are numerous methods of fixing the omentum and spleen: Rydygier's,[1] Giordano's[2] and others. An explanation of various methods may be found in Moynihan's[3] book. The most effective appears to be that of Schiassi, of Bologna, in which the omentum is fixed between the peritoneum and the muscles of the belly wall, and the sutures that fasten the spleen are probably more secure and simpler than those used in other methods. He described his method in *La semaine médicale,* May 27, 1903, and in a pamphlet, *Un nuovo trattamento del morbo del Banti* (Bologna, 1902). His operation is as follows:

FIG. 2



a, b, c, musculocutaneous incision; *d,* musculocutaneous flap; *e,* peritoneum; *f,* omentum.

1. The skin incision is made on the left side, perpendicularly to the middle of the clavicle and extending downward six to eight inches from the costal arch. At right angles to this cut, from its upper end, an incision passes to the right to a point a little beyond the median line. These two incisions are carried into the peritoneum, which is left intact at this stage of the operation. (See Fig. 1.)

2. Near the median line, and in the transverse incision, the peritoneum is picked up between two forceps and a small opening is made to let out the ascitic fluid. Then a triangular flap, composed

---

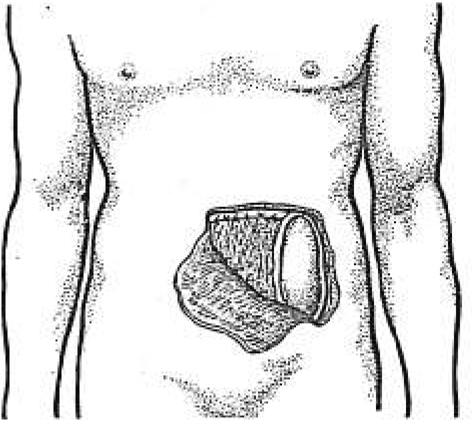
[1] Wiener klin. Wochenschr., 1895, No. 24.

[2] Rif. med., 1896, vol. i. p. 375.

[3] Abdominal Operations, Philadelphia, 1905, p. 659.

of the skin and the muscles, is dissected up, bluntly or with some use of the blade, from the peritoneum. The apex of the flap is the angle of the incisions, and the base is a line out about the region of the navel. The flap is held folded over toward the right side of the patient by hæmostats. Then the small incision made in the peritoneum to let out the ascitic fluid is enlarged horizontally, parallel to the horizontal skin incision, until two fingers may be introduced to serve as directors while the peritoneum opening is further enlarged. The omentum is to be brought out through this opening, but only about half of the peritoneal opening is made at first.

FIG. 3



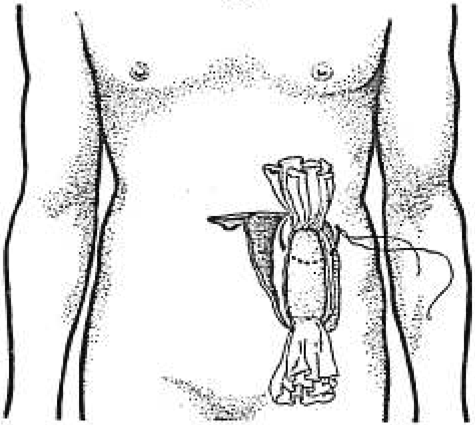
The omentum fixed and the enlarged spleen exposed.

3. The omentum is drawn out through this opening so far that its border touches the base of the everted musculocutaneous flap. The omentum is sutured to the preitoneum at the lips of the wound, serosa to serosa, the upper surface of the omentum to the upper lip and the lower surface to the lower lip, avoiding all wounding of vessels by the stitches. The incision in the peritoneum is next prolonged to the left over to a point under the apex of the costal arch, and the omentum is further stitched to the lips of the peritoneal wound as before. (See Fig. 2.)

4. The exposed omentum is next rubbed, to excite adhesion, with gauze which has been dipped into 1:1000 bichloride, spread along the bared peritoneum, and fixed along its free border to the peritoneum by a few stitches. (See Fig. 2.)

5. If splenopexy is to be done, the next step is to open the peritoneum, so as to expose the spleen, by a slit parallel to the perpendicular musculocutaneous incision, and down the entire length of this cut. (See Fig. 3.) The exposed surface of the spleen is rubbed as widely as possible with sublimate gauze to excite adhesion, as the omentum had been. Then a large piece of iodoform gauze is pushed into the space between the spleen and the diaphragm above, taking care to spread it out over the upper surface of the spleen toward the front, and this gauze is left projecting from the wound. A second large piece of gauze is inserted at the lower end of the wound so as to envelop the lower pole of the spleen, and the end of this gauze is pushed back to the bottom of the hypochondriac space. An end of the gauze is here also left projecting from the wound. (See Fig. 4.) The gauze is intended to aid in forming the juncture of the spleen with the belly wall, and to drain any seepage from the needle punctures.

FIG. 4



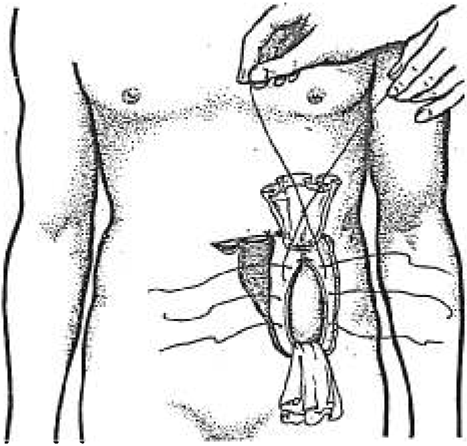
The piece of gauze above and below spleen. The needle inserted for a suture to fix the spleen. The dotted part of the needle is supposed to be in the substance of the spleen.

6. The musculocutaneous flap is now brought up over the fastened omentum. Then a semicircular needle is used, which has a diameter of from six to eight centimetres and one millimetre thickness, and heavy catgut, to fix the spleen to the belly wall. The skin is pushed aside and the needle is inserted about four centimetres to the left of the vertical musculocutaneous incision, passed through muscles and peritoneum, then into the substance of the spleen and

brought out through the preitoneum near the fixed omentum, into and through the muscles (not the skin) of the original triangular flap (see Fig. 4), so that finally the muscles along the left border of the vertical wound will join the muscles of the flap and cover the spleen. Similar interrupted sutures are inserted at two fingers' breadth distance down the vertical incision. There will be three, four or six of these sutures according to the size of the spleen. These sutures are not tied at once, but left held by suspended hæmostats. (See Fig. 5.)

7. Next the incisions in the belly wall are closed, as in any cœliotomy. The sutures begin at the angle of the wound near the gauze, and silk or heavy catgut is used. The sutures are tied as they are inserted, except near the gauze at the top and bottom of the spleen. The sutures near the gauze are left untied for a few days until after the gauze has been drawn out. As the surgeon goes down the wound he meets and draws together and ties separately the sutures that are in the spleen itself and are fastened to the suspended hæmostats. The transverse musculocutaneous wound is afterward closed as in any cœliotomy case.

FIG. 5



The sutures for fixing the spleen are in position; the upper two are tied.

An analysis of the urine is of help in determining the indications for surgical intervention in the ascites of hepatic cirrhosis. If the kidneys are unsound, of course, the prognosis becomes proportionately unfavorable. Glycosuria from an insufficiency of the hepatic cells may also affect the prognosis. Urobilinuria, too, is associated with grave conditions of hepatic cirrhosis.

Schiassi thinks the secretion of urea is closely connected with the integrity of the hepatic cells, and that a disordered condition of these cells affects the elimination of urea. Edsall[1] says that he has studied the ratio between the total nitrogen and the ammonia-nitrogen of the urine in liver disease, and he holds that this ratio has no importance in the diagnosis of hepatic pathological conditions. With Schiassi in his opinion that urea is chiefly formed by the liver are the clinical testimony of Charcot and Brouardel, the chemical experiments of Stolnikow, Schroeder and Richet, and the work of Cyon, Pawlow and Neuki.

If the ascites is connected with peritonitis care must be exercised in determining the time for operation. When the peritonitis is acute operations will almost always make the condition worse, and the prognosis becomes very grave. Again, if the ascitic fluid on tapping is heavy, or turbid, or contains much albumin; if there is elevation of temperature, pleuritic effusion or abdominal pain, surgical intervention had better be deferred. Where there is no peritonitis we should operate at once, either by fixing the omentum or the spleen and omentum according to the nature of the cirrhosis.

In Banti's disease both omentum and spleen should be fixed, and the earlier this is done the better. If the ascites is malarial in origin both epiplopexy and splenopexy should be done. If Banti's disease has been diagnosed before ascites sets in, splenopexy alone suffices. In the splenic anæmia of adults and children as distinguished from Banti's disease, Schiassi thinks splenopexy should be performed and that the same rule holds for the splenomegalic cirrhosis described by Gilbert,[2] Popoff,[3] and Rolleston.[4]

Rolleston[5] draws attention to the fact that the development of a free communication between the portal and general systemic veins can have a bad effect at times. In such a condition much of the blood that carries the products of digestion will get into the general circulation without passing through the liver, and toxic material, which normally is stopped by the liver, thus comes into contact with the tissues. A toxæmia resembling uræmia may result. In free collateral anastomosis the kidneys are injured, probably, through lack of the protective work of the liver. Besides these disadvantages the dilated venous anastomoses may rupture.

In some of Schiassi's cases a careful test of the action of the liver extracts was made. At first he gave fresh pork liver, but this brought

[1] Quoted by Musser in article on "Some Medical Aspects of the Diseases of the Gall-bladder and Gall-ducts," International Clinics, October, 1903.

[2] Soc. de biologie, May 20, 1899, p. 403; and La sem. méd., 1900, p. 154.

[3] Lecons clinique, St. Petersburg, 1896.

[4] Diseases of the Liver, p. 318.

[5] Op. cit., p. 213.

on a profuse weakening diarrhœa. Afterward he used Merck's hepatic extract in half-gram to one gram tablets. These tablets had a decided diuretic effect and they increased the output of urea. There is only 0.066 gram of urea in one of Merck's tablets, and the elimination of urea is not, therefore, a mere throwing off of the actual tablet-content, but an effect of cell-stimulation. The food was weighed, and in one case for three days during which no hepatic extract was given the quantity of urine was 1320, 1675, and 1237 c.c.; on the next three days when one-half to one gram of the extract was given, it was 1700, 1995, and 1755 c.c. The output of urea for the first three days was 78.790 grams, for the second three days, 88.016 grams; on three following days, when two to four grams of the extract were given, the quantity of urine was 1580, 1650 and 1850 c.c.; and the total output of urea was 100.746 grams.

With an intention to improve the blood he gave fresh lamb-marrow, as had been suggested by Combe, Cervesato and Caccini after experiments made between 1895 and 1900, and the effect by inference appears to be good, although he does not describe it in detail.

REPORT OF A CASE OF PARATYPHOID FEVER.[1]

By John M. Swan, M.D.,
of Philadelphia.

The patient whose case forms the basis of this report was admitted to the Philadelphia General Hospital on June 23, 1905. It was thought at first that he was suffering from typhoid fever and that a consolidated area, which was made out at the apex of his right lung, was a patch of complicating bronchopneumonia. On the second day of his hospital residence a negative serum reaction with the bacillus typhosus was reported. Two days later a positive diazo reaction was demonstrated in his urine. A blood culture made at the same time that the serum reaction was studied was reported sterile. It was then supposed that the patient might be suffering from beginning tuberculosis on account of the consolidation at the right apex; but resolution gradually occurred in this area and the cough as well as the physical signs disappeared. On the eleventh day of his hospital residence agglutination reactions were attempted between the patient's blood serum and the alpha and beta paratyphoid organisms used by Fox[23] in his agglutination studies. In a 1: 40 dilution there was what seemed to be a tendency to aggluti-

---

[1] Read at the meeting of the Section of General Medicine, College of Physicians, December 11, 1905.